



CASE REPORT

Hemisection for treatment of an advanced endodontic–periodontal lesion: a case report

H. Haueisen & D. Heidemann

Department of Restorative Dentistry, University of Dentistry ZZMK (Carolinum), Johann Wolfgang Goethe University, Frankfurt, Germany

Abstract

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Aim To emphasize the importance of primary endodontic treatment when dealing with endo–perio lesions and to demonstrate the considerable healing potential of the endodontic aspect.

Case report After several years of unsuccessful symptomatic periodontal treatment, an advanced endo–perio lesion on a right-mandibular first molar was successfully treated by root-canal treatment and hemisection after the re-evaluation of the lesion. This successful treatment appeared to have a positive effect on the patient's general well-being.

Key learning points

- The origin of a combined endo–perio lesion is indicated by its clinical and radiographic appearance. The periodontal situation is often misinterpreted.
- The prognosis for the endodontic element of treatment is excellent.
- Local pathologic processes in the oral cavity may affect a patient's general health.

Keywords: endodontic–periodontal lesion, headache, hemisection, local periodontal defect, root-canal treatment.

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Introduction

There is a close ontogenetic relationship between endodontic- and periodontal tissue structures, which is anatomically reflected in the apical foramen and accessory and lateral canals (Pineda & Kuttler 1972, Burch & Hulen 1974, Gutmann 1978, Harndt 1979, Schroeder 1987).

Correspondence: Dr Helga Haueisen, Department of Restorative Dentistry, University of Dentistry ZZMK (Carolinum), Johann Wolfgang Goethe University, Theodor-Stern-Kai 7, 60590 Frankfurt, Germany (Tel.: +49 69 63015637, fax: +49 69 63016741; e-mail: D.Heidemann@emuni-frankfurt.de).

Clinically, this relationship promotes the spread of infection, potentially resulting in typical manifestations of endo-perio osseous lesions (Nolden 1986, König *et al.* 1994, Simon & Workman 1994, Bergenholtz & Hasselgren 1997).

These lesions often remain free of symptoms for long periods, as they are rarely diagnosed until the disease starts manifesting itself in the form of acute symptoms of inflammation and/or increased pain. Sometimes, the lesions are detected accidentally during a general check-up (Simon & Workman 1994, Bergenholtz & Hasselgren 1997, Trope 1998, Haueisen *et al.* 1999). Once symptoms occur, they tend to be so severe, and the periodontal aspect can seem so dominant, that dentists tend to settle for strictly symptomatic periodontal therapy whilst overlooking the endodontic aspect. The cumulative effects of carious and iatrogenic irritation acting on the tooth/pulp often do not get the attention they deserve in the diagnostic workup, and are not recognized as a potential cause of chronic pulpitis, frequently associated by sclerosed root canals (Simon & Workman 1994, Kim & Trowbridge 1998, Haueisen *et al.* 2000). Teeth with endo-perio lesions typically have a history of deep caries treated and subsequently covered with a full or partial crown.

Endo-perio lesions are difficult to classify, because they lack the characteristic manifestations of strictly endodontic or strictly periodontal lesions (Kresic 1994, Löst 1994, Kocher 1997, Haueisen *et al.* 1999, Ratka-Krüger *et al.* 2000). Long-term preservation of the tooth seems an unlikely prospect in the presence of clinical and radiographic findings such as acute inflammation, isolated deep pockets and circumradicular/interradicular radiopacities. It is difficult to distinguish by hindsight which parts of the lesion are endodontic and which parts are periodontal in origin.

The classification by Mutschelknauss (1975), Guldener (1975) or Simon *et al.* (1972) is based on aetiology and describes the development of endo-perio lesions, whilst the classification by Geurtsen *et al.* (1985) focuses on therapeutic and prognostic aspects (Tables 1 and 2).

For the treatment of endo-perio lesions to be successful, it is helpful to understand the pathogenesis as well as the clinical and radiographic manifestations of endodontic and periodontal lesions.

Endo-perio lesions that are primarily endodontic in origin characteristically expand to the periodontal structures *via* the apical foramen, resulting in an osseous defect that progresses relatively fast along the periodontal ligament from apical to coronal, or forms a sinus tract. The probing depths of the tooth remain normal until a closely circumscribed location

Table 1 Classification according to Mutschelknauss (1975) and Guldener (1975)

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|-----|---|
| (1) | Lesions of endodontic origin with periodontal involvement |
| (a) | Expansion of the pulp lesion, either periapically or para-/interradicularly <i>via</i> the accessory and lateral canals |
| (b) | Iatrogenic: by perforation |
| (2) | Lesions of periodontal origin with endodontic involvement |
| (a) | Iatrogenic: periodontal therapy requires hemisection/apicectomy |
| (b) | Retrograde pulp infection |
| (3) | Combined endodontic-periodontal lesions where two independent defects have merged into one |
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Table 2 Classification according to Geurtsen *et al.* (1985)

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- | | |
|-----|---|
| (1) | Combined lesions requiring only a single root-canal treatment (favourable prognosis) |
| (2) | Combined lesions requiring both endodontic and periodontal treatments (less favourable prognosis) |
| (3) | Combined lesions with little hope of successful treatment (poor prognosis) |
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reveals significant probing depths of 10–12 mm. Radiographs show a normal-shaped alveolar ridge in the proximal area and osteolysis spreading from an apical/interradicular focus to the periapical area (König *et al.* 1994, Kresic 1994).

Periodontal structures such as the attachment of Sharpey's fibres in the (still healthy) root cementum initially remain intact. This means that the bony defect may fully regenerate after the inflammatory focus is removed by endodontic treatment. Only if the lesion has persisted for an extended period will the epithelial tissue migrate into the periodontal pocket and result in a combined lesion that, much like premature scaling of the root surface, reduces the potential for complete healing (Nolden 1986, König *et al.* 1994, Löst 1994, Simon & Worksman 1994).

The periodontal aspect of combined lesions develops over an extended period of time, starting at the marginal periodontal tissue. Radiographs reveal a coronally wide vertical bony defect. Closed or open surgical periodontal procedures will usually only achieve healing by repair; in some indications, guided tissue regeneration (GTR) yields better results (Cortellini *et al.* 1996).

To structure the complex treatment of endo-perio lesions, a treatment concept was developed by Haueisen *et al.* (1999) that combines endodontic and periodontal measures in a special sequence and at defined intervals (Fig. 1). The different progression of lesions of endodontic *versus* periodontal origin (relatively fast/slow development of bony defects), as well as the different levels of healing (regeneration/repair), were taken into account.

Following initial treatment, priority is given to the endodontic aspect to allow healing by regeneration—unless the patient presents with acute periodontal symptoms. Any decision in favour of periodontal measures or even surgical endodontics will be based on the clinical and radiographic findings 6 months after root-canal therapy. An additional 6 months later, the regeneration of the endodontically induced part of the bony defect should be more or less complete; any residual periodontal defects should subsequently be treated by regenerative periodontal techniques. In most cases, it takes 18 months after the completion of root-canal treatment before it can be definitively determined whether or not the treatment as a whole was successful.

The objective of the following case report is to present both the characteristic diagnostic features of an endo-perio lesion and a treatment concept that can be applied even in complex cases.

During the course of successful treatment, the patient reported improvement in her general state of well-being, including the disappearance of a long-standing unilateral headache.

Case report

A 62-year-old woman presented in April 2000 to inquire about options for preserving tooth 46 (Figs 2 and 3). The tooth was characterized by gingival reddening and swelling at its distobuccal aspect. The patient complained of periodic discharge of pus from the periodontal pocket, sensitivity on percussion, tooth mobility, and intermittent pain.

Radiographs from 1994 documented a bony defect around tooth 46, which continuously expanded over the years (Fig. 4 a–c). The patient suspected a special predisposition to disease because she had been diagnosed with polyarthritis and osteoporosis in 1980, which together with a history of injuries had already required surgical procedures in two joints. Several degenerative phenomena along the spinal cord were also recorded. The patient herself complained of poor metabolic disposition and/or impaired immune defence mechanisms resulting from a severe disease of unknown aetiology in early childhood as well as malnutrition in childhood and adolescence (1946–54). She regularly took minerals, vitamins, and enzymes to compensate for these reported deficiencies.

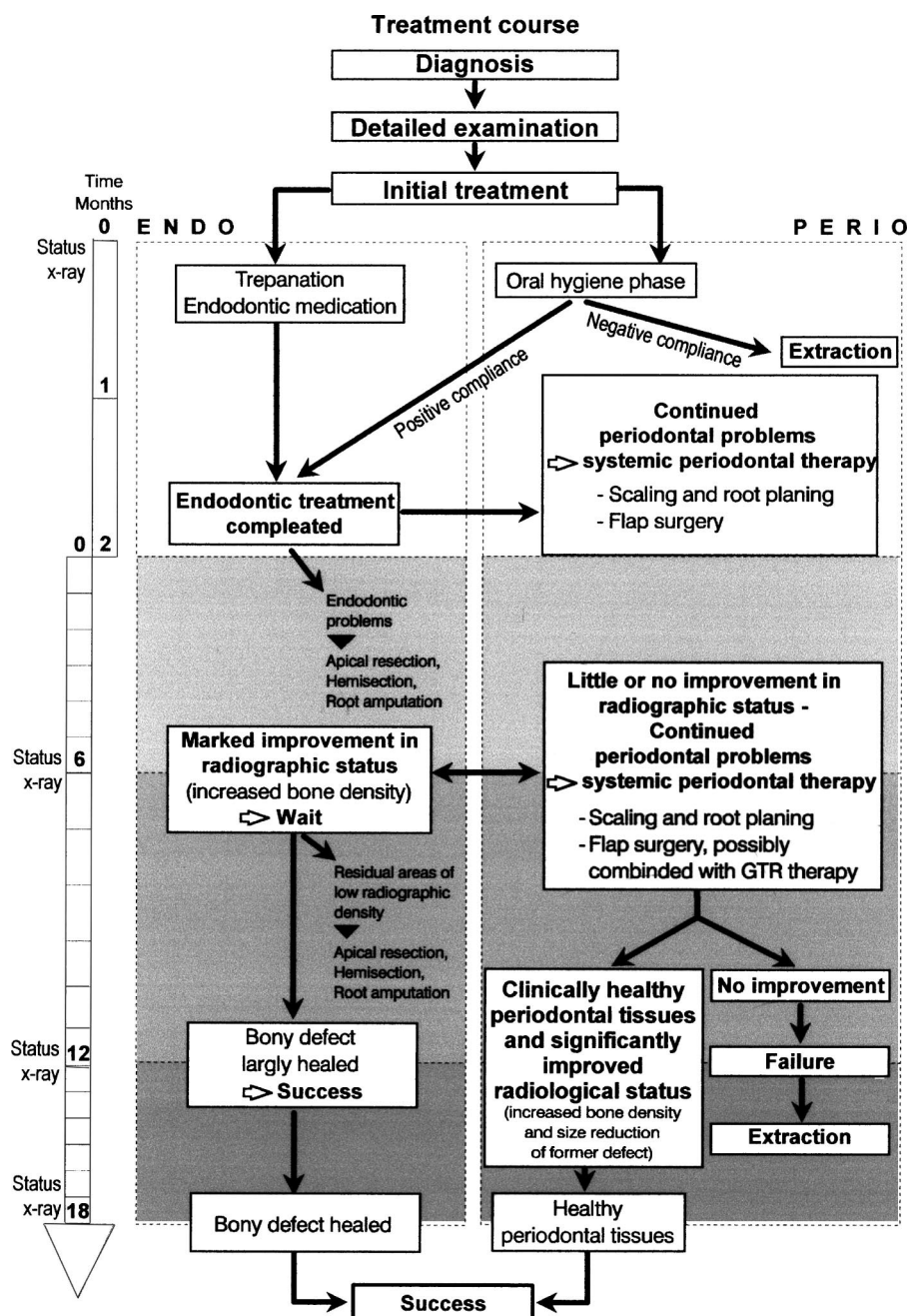


Figure 1 Treatment concept for combined endodontic-periodontal lesions.

The patient also mentioned stress-dependent headaches dating back to an accident-related head contusion. In addition, she had been afflicted with unilateral stress-independent headache attacks at a more distal cranial location for 2 or 3 years, which were becoming increasingly frequent and intense. She was considering seeing a neurologist and wanted to find out whether these attacks might be connected with the dental lesion. Likewise, the patient showed erythrocyte-sedimentation rates near the upper tolerance limit for a number of years without any other clinical phenomena to match this finding.



Figure 2 Adequate restorations (partial gold crown/inlay) on tooth 46/47.

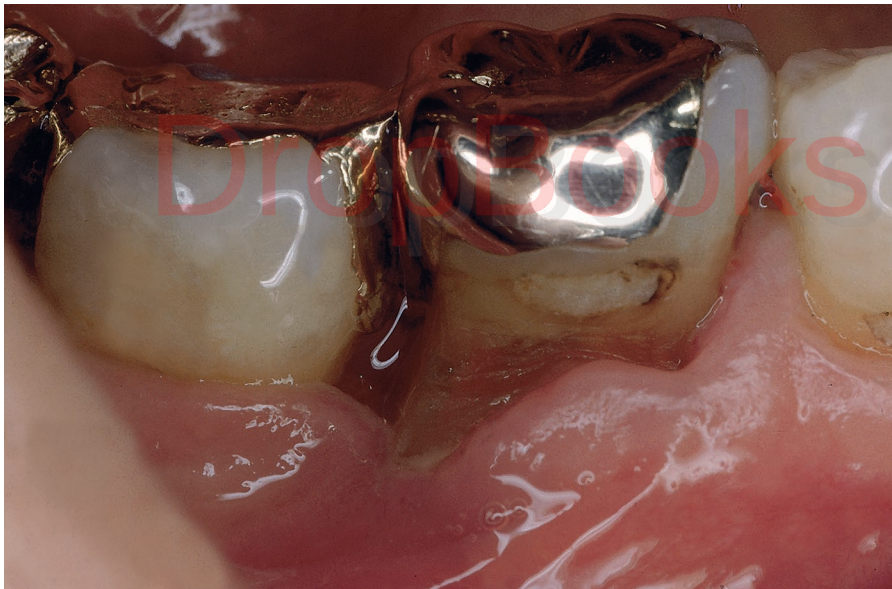


Figure 3 Recession in the area of the distal vestibular root of tooth 46 involving loss of the interdental papilla.

Dental history

Around the year 1991, tooth 46 had been restored with a partial gold crown. A year later, it had caused non-specific symptoms, which were managed by local periodontal treatment and short-wave therapy. When symptoms recurred 3 years later, the patient decided to change her dentist. A panoramic tomograph (DPT) and a single-tooth radiograph of region

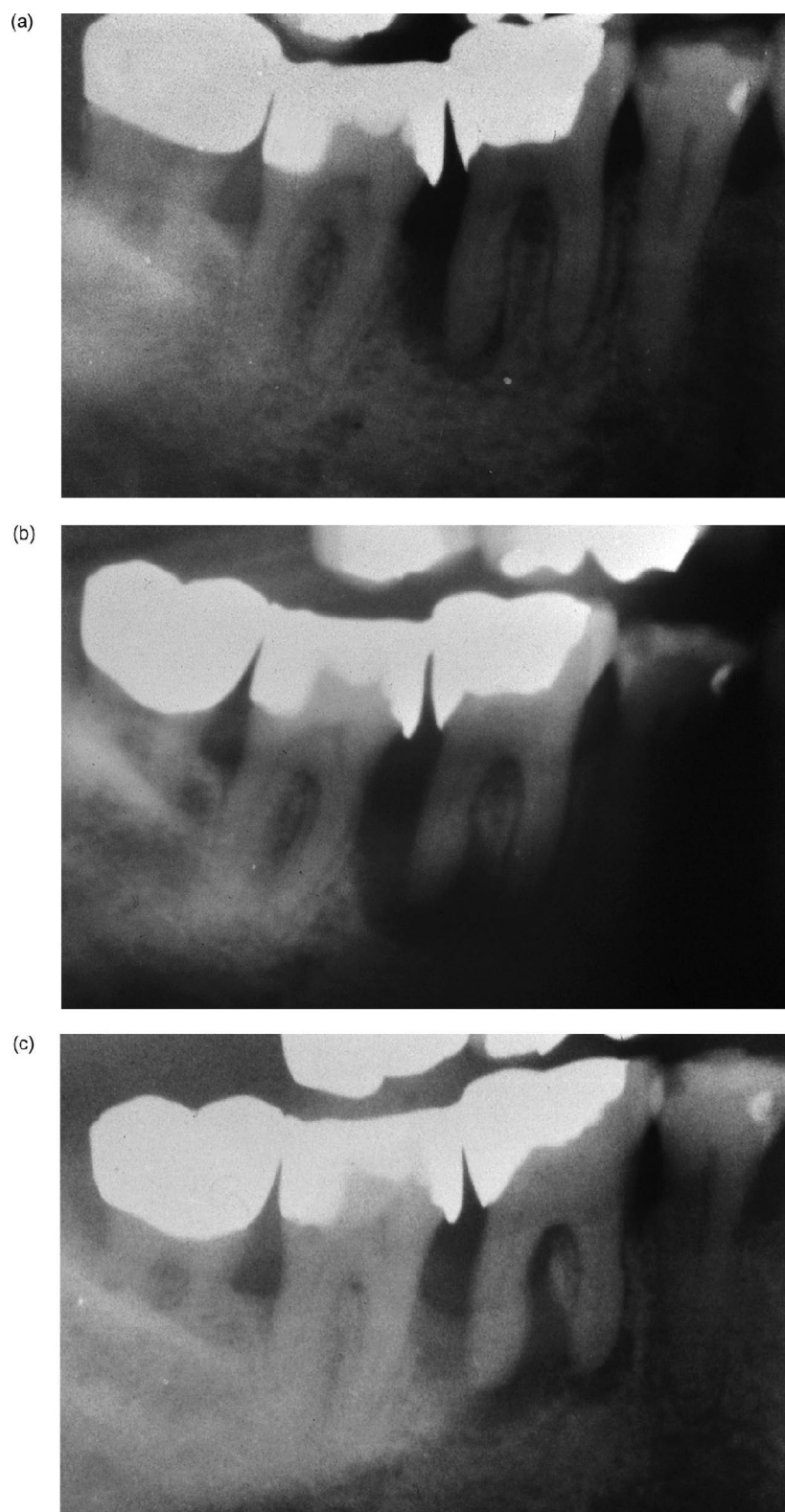


Figure 4 Region 46 on DTPs obtained in 12/94 (a), 12/96 (b) and 3/00 (c).

46 revealed a mild-furcal involvement and circumradicular radiopacity around the distal root, as an almost complete loss of the interdental septum of teeth 46/47.

Over the next few months, the dentist treated the affected site nine times with a corticosteroid ointment. At first, the patient was largely free of symptoms. A DPT obtained at a routine dental check-up in late 1996, revealed that the bony defect in region 46 had expanded further, but no treatment had been performed. At the next routine examinations in 1998 and 1999, the patient had mild complaints. The situation deteriorated in early 2000, with intermittent pain, local swelling, and increased discharge of pus from the periodontal pocket of tooth 46. Over the next 2 weeks the affected site was treated six times with metronidazole gel, and another DPT was taken, which showed that the bony defect had expanded even further. The dentist now performed flap surgery for subgingival calculus removal, and root planing in an attempt to preserve the tooth. Extraction of the tooth was also discussed as an alternative.

In April 2000, the situation in region 46 was characterized by a negative pulp-vitality test, and a positive percussion test. The distal gingival tissues were reddened and swollen. There was a distal vestibular recession of 4 mm, and circumferential probing depths in the area of the distal root were 8–12 mm (Fig. 5), classes II and III tooth mobility, and the furcation was probable throughout (grade III furcal lesion). A standard single-tooth radiograph obtained to achieve a more detailed view revealed the extensive circumradicular bone loss predominantly in the distal root area, the mesial root still showing interradicular bone structures in its upper-third and mesioproximal bone structures over two-thirds of the root length (Fig. 15a).

The patient exhibited good oral hygiene and no missing teeth. Apart from tooth 46, the periodontal situation (DPT taken on March 2000) was normal and there were no clinical signs of gingivitis.

Presumptive diagnosis

The presence of an advanced endo-perio lesion was suggested by the following findings from the dental history and the clinical and radiographic examination:

- Negative pulp-vitality test;



Figure 5 Significantly increased probing depth on the distal vestibular segment of tooth 46.

- Periodontal defect characterized by deep local probing depths in the distal root circumference;
- Localized defect in a patient with otherwise healthy periodontium;
- Radiographic follow-up in 1994, 1996, and 2000;
- Moderate pain, non-specific complaints, bite sensitivity, recurrent exudation; and
- Failure of previous periodontal therapy.

Therapy

It was decided that, instead of extracting tooth 46, an attempt should be made to partially preserve it by hemisection, with removal of its distal half followed by restoring teeth 46 and 47 with splinted crowns. The definitive decision had to be made perioperatively, once it was known how much bony substance was left in the furcation area. Total preservation was not considered a realistic option, because the osteolysis in the distal segment was so far advanced and the root had already been scaled several times. Periodontal regeneration was considered unlikely.

The patient consented to our proposed treatment plan after being comprehensively informed about the methods and risks of the treatment, and how many radiographs would have to be obtained. The partial crown on tooth 46 was removed and the pulp chamber opened. The mesial root canals were shown to be almost completely obliterated and had to be instrumented using ISO size 06 root-canal instruments (Maillefer, Ballaigues, Switzerland) and a Canal FinderTM device (S.E.T., Emmering, Germany) (Figs 6 and 7). Following placement of a calcium hydroxide paste, a compomer restoration (Compoglass[®], Vivadent, Schaan, Lichtenstein) was placed to close the proximal defects.

The root-canal treatment was continued using rubber dam and intensive irrigation. The root-canal filling was introduced by lateral condensation with gutta-percha and Roeko Seal (Roeko, Langenau, Germany).

Hemisection was performed 3 weeks later. Owing to the high mobility of the tooth 46, a temporary restoration was planned to stabilize the mesial root postoperatively. Following

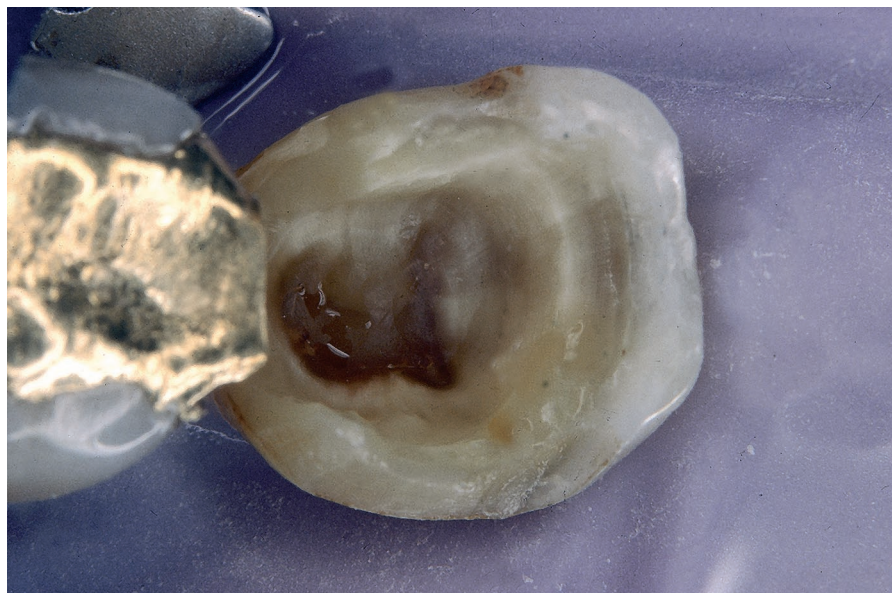


Figure 6 Endodontic cavity in tooth 46 with almost completely obliterated root-canal entrances.

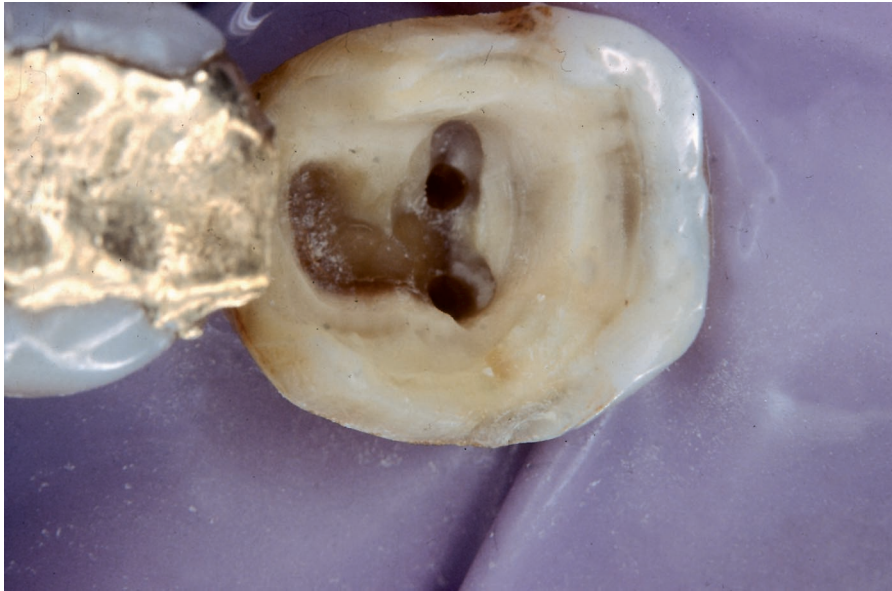


Figure 7 Endodontic cavity in tooth 46 after opening both mesial root canals.

anaesthesia, tooth 47 was restored with layered compomer (Figs 8–11). Teeth 46/47 were prepared to receive splinted temporary crowns. Only then the tooth 46 was dissected, the distal root extracted, and the wound was curetted. On probing the root stump, the distal bony margin could be felt 1 mm below the level of the gingiva. After placing sutures to adapt the wound margins, the temporary restoration was re-lined with composite, cut back to fully expose the interdental space, and inserted.



Figure 8 Teeth 46/47 after adaptations made to the restorations and placement of a layered compomer filling.



Figure 9 Preparation for teeth 46/47 to receive splinted crowns.



Figure 10 Dissection of tooth 46 at the gingival level.

Healing was uneventful. A temporary restoration was inserted in week 5 postoperatively after assessing the mesial mobility of tooth 46 (grade I) and circum-radicular probing depths (Figs 12–14).

The patient reported that her attacks of unilateral headaches had been significantly reduced during endodontic treatment, and did not re-occur following a final severe attack



Figure 11 Distal root of tooth 46 after extraction.

the day after the hemisection. The patient's subjective general health was significantly improved. The erythrocyte sedimentation rates fell from near the upper threshold levels (e.g. 16/32) back to normal (10/20).

Clinical and radiographic follow-ups 6 and 12 months postoperatively showed Grade 0–I and I tooth mobility and a probing depth of 1–3 mm for the mesial root of tooth 46



Figure 12 Normal probing depths at tooth 46 5 weeks after the procedure.



Figure 13 Laboratory-made temporary restoration on teeth 46/47.

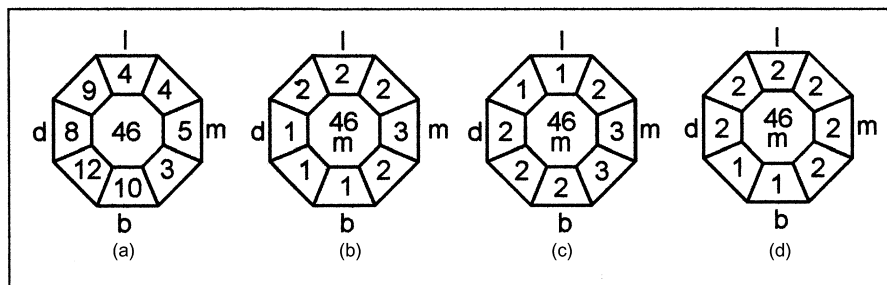


Figure 14 Probing depths at tooth 46 in April (a), June (b), December (c) of 2000, and June of 2001 (d).

(Fig. 14 a–d). The radiographs showed that the bone density between 46 and 47 had continuously increased and the original defect levelled out (Fig. 15 a–d).

Future follow-up examinations will determine when the definitive restoration (presumably a bridge between 46 mesial and 47) can be inserted.

Discussion

Treatment procedure

Successful treatment of endo-perio lesions depends upon their timely and accurate diagnosis. The patient had been treated for unspecific symptoms as far back as 1991. A chronic pulpitis developed to necrosis and 3 years later induced an endo-perio lesion that had not been recognized as such. Rather, the finding of significant probing depths and a radiographically visible bone loss were interpreted and treated as a periodontal defect, the treatment of which temporarily rendered the patient free of symptoms.

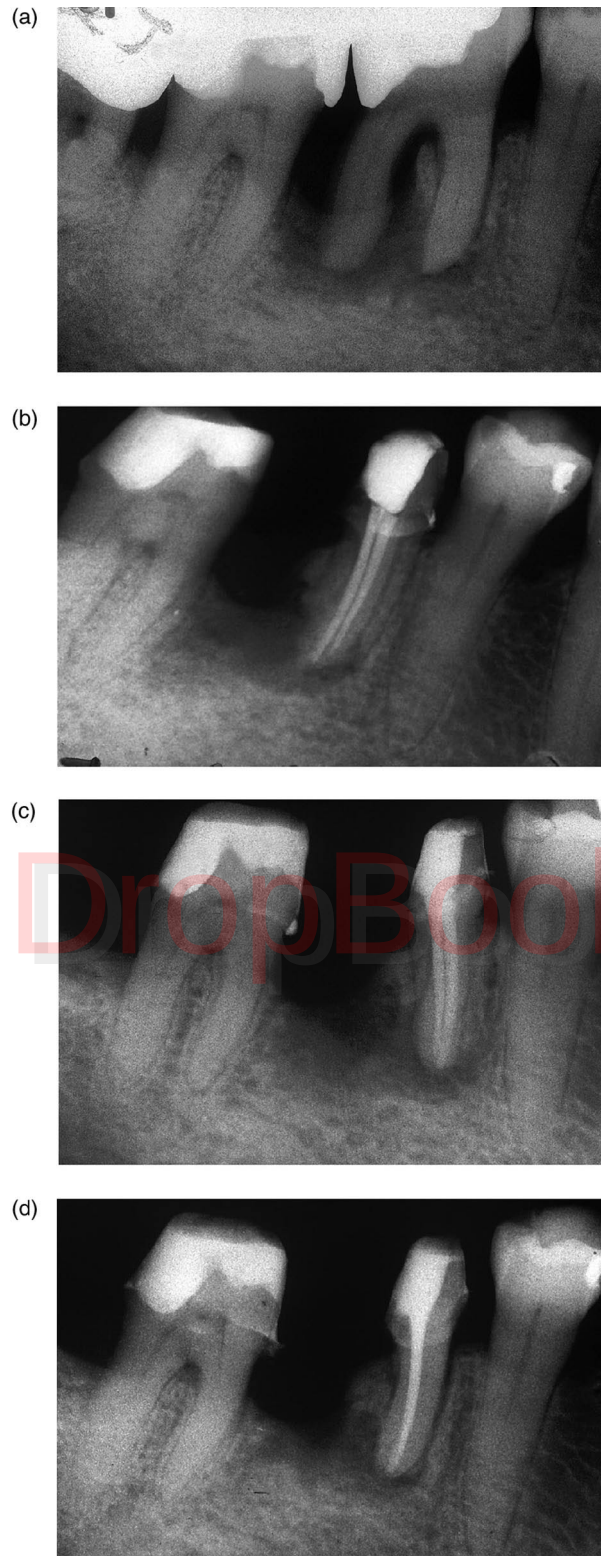


Figure 15 X-ray image of bony defect at tooth 46 in April (a), June (b), December (c) of 2000, and June of 2001 (d).

A thorough differential diagnosis would have made it apparent that the manifestations of the defect were atypical. The periodontal defect was confined to a single tooth. It extended in an arch-like fashion from the apical area along the distal root surface rather than being angular. Based on this finding, at least the possibility of a retrograde pulpitis should have been considered (Geurtsen *et al.* 1985, König *et al.* 1994, Simon & Worksman 1994, Bergenholtz & Hasselgren 1997). The vitality test is not an absolute but a relative indicator of pulpal integrity. Especially in teeth with multiple roots, the situation may be misinterpreted by a variable degree of pulp degeneration in different tooth segments or root canals (Löst 1994).

The fact that the DPT of 1996 demonstrated progression of the bony defect would have been another indicator of an endodontic lesion. It is also known that cumulative carious and iatrogenic irritation can initiate pulp necrosis.

In early 2000, when the symptoms once again became acute and the clinical and radiographic findings had acquired significant dimensions, the patient received drug therapy and surgical periodontal treatment. Despite the fact that it would have been high time to refer the patient to a specialized unit, there was no reconsideration of the diagnosis, and the treatment once again failed.

When the patient first presented at our clinic, this treatment history called for a thorough diagnostic workup. Several factors played a determining role in evaluating the current status of the endo-perio lesion as the patient presented at our clinic, in order to determine the subsequent course of therapy. The patient's dental history and the radiographs of 1994 showed that the endo-perio lesion of tooth 46 was primary endodontic in origin.

Lesions of a purely endodontic origin, like the endodontic aspects of combined lesions, have an excellent prognosis. This was demonstrated by a number of case reports (Simon & Worksman 1994, Bergenholtz & Hasselgren 1997, Trope 1998) and by a study of 10 lower molars conducted by Haueisen *et al.* (2000). Endodontic treatment alone results in complete reversion in many cases.

Our case was complicated by the fact that the lesion had existed for several years. An initially minor bony defect now showed a sizeable aperture coronally. There was a high probability that the junctional epithelium had proliferated downward, resulting in a combined endo-perio lesion with secondary periodontal involvement, typically associated with a less favourable prognosis.

From a clinical viewpoint, the attempt to preserve the mesial root was justified because probing depths in the mesial circumference of the tooth were only slightly increased. Radiographically it was justified by an intact gingival septum between teeth 46/45 and interradicular bony structures in the coronal-third of the mesial root. Also, the single-tooth radiograph showed fine bony structures mesially in tooth 47 at two-thirds of the root.

Depending on intraoperative findings, guided tissue regeneration could also be considered as an alternative. The intraoperative situation was such, however, that the procedure carried out appeared sufficient, a decision later confirmed by the favourable postoperative outcome.

Patient's general health

Another interesting aspect is the apparent improvement in the patient's general health. The unilateral stress-independent headache attacks continuously decreased and finally vanished as the healing of the defect proceeded.

Focal infection theory was prominent during the early 20th century, but scientific evidence was lacking (Murray & Saunders 2000). In the present case, erythrocyte sedimentation rates returned to normal after treatment, which is plausible given the elimination of the chronic infection around tooth 46. What is more difficult to explain is why the

unilateral headache disappeared. Pertinent publications are rare. There are only a few case reports, none of them too well documented. A systematic follow-up by Lunardon & Barolin (1997) showed that in one-quarter of the 785 headache patients odontogenic factors were implicated. Of these, 158 were systematically followed. Two-thirds of these showed improvement of the headache symptoms. Suitable treatment of oral foci is recommended, although improvement does not necessarily follow.

General health – local microbial findings

The issue of intraoral pathological findings as a factor in systemic health problems is reopened as the development of improved microbiological techniques and evaluation methods continues. C-reactive protein serum levels are acknowledged to be clinical indicator of microbiological findings. Noack *et al.* (2001) proved the relevance of CRP serum levels following periodontitis treatment in a study that included 174 patients (65 healthy patients, 59 with mild periodontitis, 50 with chronic periodontitis). Ebersole *et al.* (1997) examined the course of C-reactive protein and haptoglobin serum levels following treatment of adult periodontitis (AP $n = 40$; control group $n = 35$).

Crucial data have been collected in the field of periodontology, where chronic periodontitis is considered a risk factor for cardiovascular disease, atherosclerosis, bacterial pneumonia, and premature births (Debelian *et al.* 1994, DeBowes 1998, Murray & Saunders 2000, Garcia *et al.* 2001). The Gram-negative bacteria seem to be particularly important in this context in that they cause secondary systemic effects – formation of endotoxins, cytokines, and inflammation mediators.

To what extent latent endodontic processes—along with haematogenic infection (e.g. risk of endocarditis)—have a systemic impact needs to be elucidated by further studies. For example, an influence on rheumatoid arthritis is under consideration. Infected endodontic tissue has been shown to contain a high percentage of the Gram-negative bacteria (Debelian *et al.* 1994, Murray & Saunders 2000, Garcia *et al.* 2001), so that secondary effects *via* endotoxins and inflammation mediators appear possible.

To summarize, it would certainly be useful to collect case reports and clinical studies and to conduct endodontological intervention studies to improve our current understanding of how oral microorganisms are linked to systemic health.

Conclusions

For the endo-perio lesions to be treated successfully, an accurate diagnosis is mandatory. This diagnosis must cover both the endodontic and the periodontal component of the lesion. Where the primary aspect cannot be evaluated, endodontic treatment should be given precedence, followed by a wait-and-see approach until a decision for any additional endosurgical and/or periodontal procedure can be focussed. Further studies are required to elucidate the relationship between common microbe induced inflammatory conditions (marginal and apical periodontitis) and systemic health.

References

- Bergenholtz G, Hasselgren G (1997) Endodontics and periodontics. In: Lindhe J, ed. *Clinical Periodontology and Implant Dentistry*, 3rd edn. pp. 296–331. Copenhagen, Denmark: Munksgaard.
- Burch JG, Hulen ST (1974) A study of the presence of accessory foramina and the topography of molar furcations. *Oral Surgery, Oral Medicine and Oral Pathology* **38**, 451–5.
- Cortellini P, Pini-Prato G, Tonetti MS (1996) Periodontal regeneration of human intrabony defects with bioresorbable membranes. A controlled clinical trial. *Journal of Periodontology* **67**, 217–23.

- Debelian GJ, Olsen I, Tronstad L (1994) Systemic diseases caused by oral microorganisms. *Endodontics and Dental Traumatology* **10**, 57–65.
- DeBowes LJ (1998) The effects of dental disease on systemic disease. *Veterinary Clinics of North America Small Animal Practice* **28**, 1057–62.
- Ebersole JL, Machen MJ, Willmann ST, DE (1997) Systemic acute-phase reactants, C-reactive protein and haptoglobin, in adult periodontitis. *Clinical and Experimental Immunology* **107**, 347–52.
- Garcia RI, Henshaw MM, Krall EA (2001) Relationship between periodontal disease and systemic health. *Periodontology* 2000 **25**, 21–36.
- Geurtsen W, Ehrmann EH, Löst C (1985) Die kombinierte endodontal–parodontale Erkrankung. *Deutsche Zahnärztliche Zeitschrift* **40**, 817–22.
- Guldener PHA (1975) Die Beziehung zwischen Pulpa- und Parodontalerkrankungen. *Deutsche Zahnärztliche Zeitschrift* **30**, 377–9.
- Gutmann JL (1978) Prevalence, location and patency of accessory canals in the furcation region of permanent molars. *Journal of Periodontology* **49**, 21–6.
- Harndt R (1979) Beziehungen zwischen Endodont und Parodont. *Deutsche Zahnärztliche Zeitschrift* **34**, 453–5.
- Haueisen H, Ratka-Krüger P, Heidemann D (1999) Paro–Endo-Läsion: Unerwartete Ursache parodontaler Destruktion. In: Heidemann D, ed. *Deutscher Zahnärztekalendar*, pp. 97–129. München, Germany: Hanser Verlag.
- Haueisen H, Ratka-Krüger P, Heidemann D (2000) Knochenregeneration induziert durch endodontische Behandlung bei endodontal-parodontalen Läsionen unterer Molaren. *Deutsche Zahnärztliche Zeitschrift* **55**, 192–6.
- Kim S, Trowbridge HO (1998) Pupal reactions to caries and dental procedures. In: Cohen ST, Burns RC, eds. *Pathways of the Pulp*, 7th edn. pp. 531–51. St. Louis, USA: Mosby.
- Kocher T (1997) Parodont und Endodont. In: Heidemann D, ed. *Praxis der Zahnheilkunde*. Bd. 4 (Parodontologie), 3rd edn. pp. 305–12.
- König J, Kocher T, Plagmann H-CH (1994) Wechselwirkung zwischen Parodontitis und Pulpitis und ihre Auswirkung auf die Therapie bei kombinierten endodontal/parodontalen Läsionen. *Parodontologie* **2**, 93–102.
- Kresic T (1994) Therapie der kombinierten Paro–Endo-Läsion. *Endodontie* **3**, 233–8.
- Löst C (1994) Differentialdiagnostik bei Erkrankungen des Parodonts/der Pulpa. *Deutsche Zahnärztliche Zeitschrift* **49**, 301–6.
- Lunardon M, Barolin GS (1997) Odontogenic (concomitant) aetiology of headache. *Wiener Medizinische Wochenschrift* **15**, 365–8.
- Murray CA, Saunders WP (2000) Root canal treatment and general health: a review of literature. *International Endodontic Journal* **33**, 1–18.
- Mutschelknauss R (1975) Endodontie in der Parodontologie. *Deutsche Zahnärztliche Zeitschrift* **30**, 372–6.
- Noack B, Denardin E, Genco RJ, Hoffmann Th (2001) Systemische Akutphaseantwort bei Parodontitis marginalis. *Deutsche Zahnärztliche Zeitschrift* **56**, 122–5.
- Nolden R (1986) Parodontologische Aspekte bei endodontischen Maßnahmen. *Deutsche Zahnärztliche Zeitschrift* **41**, 906–12.
- Pineda F, Kuttler Y (1972) Mesiodistal and buccolingual Roentgenographic investigation of 7,275 root canals. *Oral Surgery Oral Medicine and Oral Pathology* **33**, 101–10.
- Ratka-Krüger P, Haueisen H, Schacher B, Neukrantz E (2000) Verlust endodontaler Integrität als Ursache für lokalisierte parodontale Läsionen. *Deutsche Zahnärztliche Zeitschrift* **55**, 417–20.
- Schroeder HE (1987) *Orale Strukturbilogie*, 2nd edn. Stuttgart, Germany: Thieme-Verlag.
- Simon HJ, Glick DH, Frank AL (1972) The relationship of endodontic–periodontic lesions. *Journal of Periodontology* **43**, 202–8.
- Simon HJ, Worksman LA (1994) Endodontic–periodontal relations. In: Cohen ST, Burns RC *Pathways of the Pulp*, 6th edn. pp. 513–30. St. Louis, USA: Mosby.
- Trope M (1998) Endodontic–periodontal interrelationships. *Alpha Omegan* **4**, 49–55.